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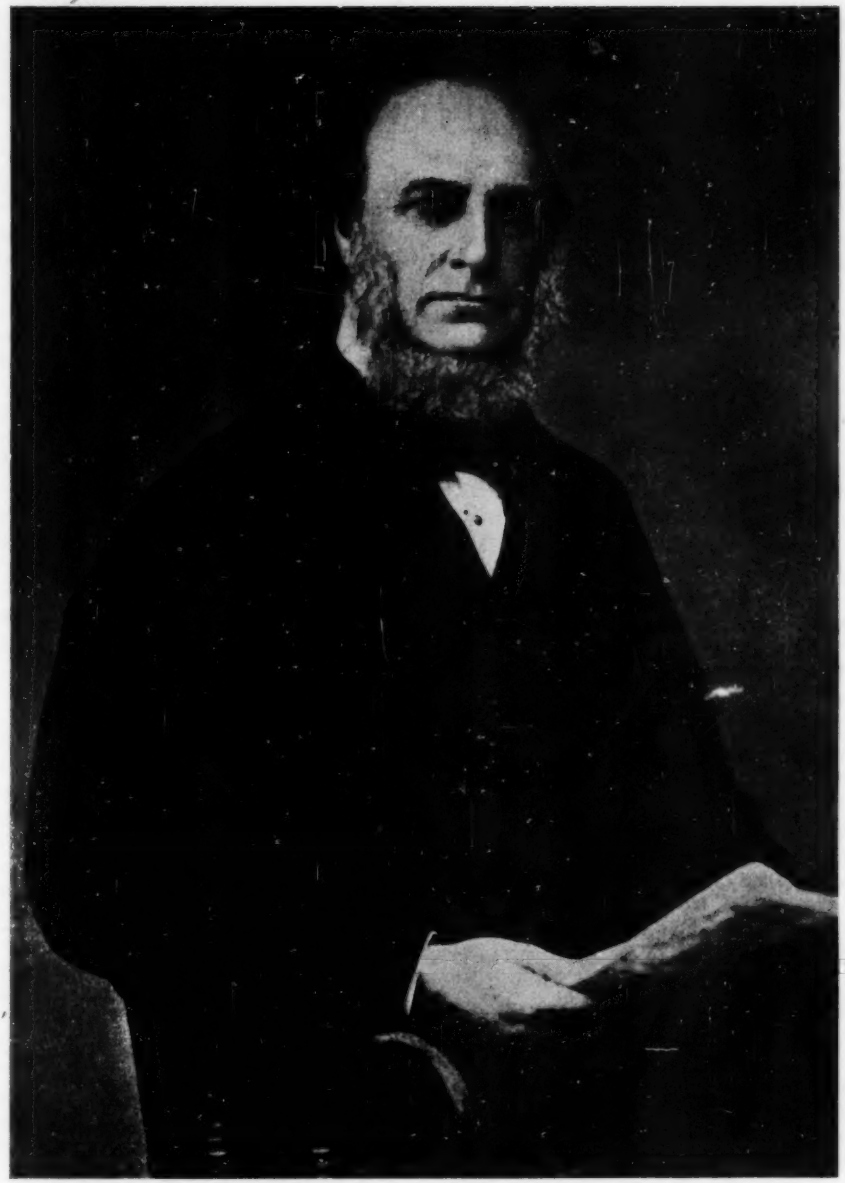

Dr. W. J. LITTLE

From a portrait in the possession of the Institute of Orthopædics (Royal National Orthopædic Hospital), University of London.

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INTRODUCTION

BY PUBLISHING a series of *Bulletins*, of which this is an atypical prototype, under the general direction of its Medical Advisory Committee, the National Spastics Society hopes to stimulate interest in the scientific and clinical aspects of the various forms of cerebral palsy. The *Bulletins* will appear four times a year, with occasional special numbers when suitable material presents itself. This introductory number forms an historical basis for the series; the second will aim at summarising the more important publications on cerebral palsy of the last year or so, in abstracts, annotations, and book reviews, with some authoritative new work. Thereafter each number will be mainly devoted to one side of the cerebral palsy problem, or be mainly addressed to one group of potential readers.

The National Spastics Society owes its origin, partly at any rate, to the anger of young parents—angry because for some obscure reasons their children were born to be handicapped and the national resources for mitigating such handicaps seemed inadequate. As the Society's work developed, research was given a place of high priority, and now a trust is being formed to administer a research fund which can thus be independent of the Society's other activities. This arrangement will simplify the planning of programmes of work under the eye of the Medical Advisory Committee, a small body that meets every month to advise on projects and approve research activities, for which a Research Physician assumes a general guiding responsibility.

Broadly speaking, the righteous anger of the founders indicates the lines on which investigation must logically proceed. "Why does cerebral palsy happen?" is still an open question if the questioner presses for details beyond the assertions of lack of oxygen at birth or a congenitally abnormal brain. Nearly a century ago DR. W. J. LITTLE put forward a theory of obstetric difficulties which was so obviously the beginning of scientific interest in the whole subject that it seemed right to start this series with his portrait and a brief note on his life, followed by an unabridged reproduction of his original article. In the interval there have been remarkably few precise additions to LITTLE's views, and one of the most urgently needed research activities is a valid prospective survey of the relation of birth trauma in its widest sense to cerebral palsy. Several adjuvant investigations are already planned or on the way, but the basic research presents a big complicated problem yet to be faced.

On the second reason for the Society's existence it is fair to make two comments. Firstly, the record of education authorities in Great Britain in caring for the handicapped child is unsurpassed anywhere. If the cerebral palsy child has often seemed to get a poor chance it may well be that in the past his intellectual attainments were overshadowed by his physical disabilities, especially by his lateness in walking and his poverty of speech and manual dexterity. On the other hand, it is unfair to conceal from the parents of such children that a proportion of them must face mental handicaps as well as physical troubles, and no amount of education—in its broadest sense—can supply the needs of the most severely retarded. The assessment of a handicapped child's mental potentialities is a difficult and responsible task.

Its importance at the very root of the problem of what to do for a child with

cerebral palsy led the Medical Advisory Committee to put forward a scheme for educational assessment recently published in booklet form. Physical assessment is still to some extent befogged by nomenclature but there are hopes that agreement can be reached on essential terminology. This must precede rational treatment, for although mechanical aids and orthopædic surgery, plus specialised physiotherapy, can do something to help, the future lies with the ability of drugs and other more radical procedures to relax hypertonic muscles and control the athetoid's movements. It is hoped from time to time to summarise the results of investigations on these lines as well as to fertilize the workings of the minds of those engaged in the wide fields where the harvest may provide something for comfort if not for cure. An authoritative Consultant Research Committee, with experts on subjects ranging from genetics to electroencephalography, meets twice a year to reduce the number of stones left unturned.

In the long run, the parents themselves have the main task to perform in helping their children, and, though these *Bulletins* are not meant primarily for them, they may show that their needs and hopes are not ignored. Some progress has been made since LITTLE's time, when, in discussing his communication, a speaker could seriously assert that the main cause of the congenitally paralysed child's handicap was teething! (See p. 35). But the great interest in the whole subject that has arisen in the last decade has not produced so much constructive work as could be wished. There are signs that more rapid progress is only a short way ahead, and it is the policy of the Society to ensure that research is not held back by lack of financial aid.

WILLIAM JOHN LITTLE, 1810-94

A Brief Biography

Originally, the Littles came from the village of Carbrook, in Norfolk, where William's grandfather was parish clerk. His father, John Little, went to live in London and became landlord of the famous Red Lion Inn, at the corner of Leman Street, Aldgate, once a favourite haunt of Dick Turpin and his highwaymen colleagues. William was born at the Red Lion on August 7, 1810. He was a delicate child and had numerous infantile ailments, one of them almost certainly being poliomyelitis, which affected the muscles of his left leg and led to a talipes equinovarus. He went to school at St. Margaret's Bay, near Dover, and having revealed a special aptitude for French was moved at the age of 13 to the celebrated Jesuit College of St. Omer, where he did very well.

When he was 16, William Little made up his mind to study medicine, so that he could learn what was known about deformities like his own and could search for a cure for them. Thus he joined that worthy band of medical men and women who have contributed to the general pool of knowledge of their own ailments. As was usual in those days, he started as an apprentice, working for two years under a local surgeon, James Sequeira. He then did three years at the London Hospital, with lectures at other hospitals as well, and at 21 he qualified as a licentiate of the Society of Apothecaries of London. In 1832 there was a series of outbreaks of Asiatic cholera in England, and Little assisted Dr. Cobb, a London Hospital physician, in investigating an outbreak in Newcastle. He was one of the first to treat cholera with saline injections, and 20 years later he turned this early experience to good account in his presidential address to the Hunterian Society on the history of intravenous therapy.

His ambition to find the cause and cure of his own disability stayed with Little when he started practice in the City of London. Having applied unsuccessfully for an appointment on the surgical staff of the London Hospital, he decided to visit Berlin and other German centres to study the work they were doing on club-foot. The dissection of numerous deformed feet convinced him that his condition arose not from any inherent defect of bone growth but from a disorder of neuromuscular co-ordination; and, furthermore, that it was amenable to surgical treatment. He had already read about the new technique of subcutaneous tenotomy of the Achilles tendon, lately devised by Louis Stromeyer, a famous surgeon of Hanover, so he sought Stromeyer's advice and help. Stromeyer agreed to operate on Little's leg and did one of his subcutaneous tenotomies on the tendo Achillis with great success. The result was a complete cure of the disability. After perfecting himself in the technique of this operation, Little returned to Berlin, where he got an M.D. for his thesis on club-foot. The professor of surgery, J. F. Dieffenbach, suggested some improvements in subcutaneous tenotomy and Little developed it still further. By 1837, when he came back to England, Little had made himself the foremost authority on club-foot and its treatment—at the age of 27!

On February 20, 1838, Little did his first tenotomy in London. Then in the following year he was elected an assistant *physician* to the London Hospital. By exchanging the status of surgeon for that of physician in this way he put himself in a somewhat anomalous position, but the fact that it was almost unheard of for a hospital physician to operate did not deter him from doing tenotomies on many cases of club-foot. He was the first to perform tenotomy on the anterior tibial tendon, for Stromeyer confined his work to the tendo Achillis. At first Little did not operate on congenital cases until the children were old enough to walk, but later he operated within a few weeks of birth.

It seems odd that the pioneer of orthopædic surgery in England was a physician to the London Hospital, though Little was only exemplifying the dictum that a surgeon is "a physician who is compelled to work with his hands". Moreover, he soon extended his efforts on behalf of cripples far beyond his own operative work. He was mainly responsible for organising a systematic attack on the whole problem of their treatment. With this aim, in 1838 he founded the Orthopædic Institution in Bloomsbury, which was so heavily besieged by patients that it had soon to be moved to larger premises in Hanover Square. In 1845 the Institution was granted a Royal Charter, and it later amalgamated with others to form what is now the Royal National Orthopædic Hospital in Great Portland Street, London University's main centre of orthopædic postgraduate teaching and research, as the "Institute of Orthopædics".

Allied to Little's work on club-foot was his description of congenital spastic diplegia, or cerebral palsy, since known as "Little's disease". He first described what he called spastic rigidity of the limbs of newborn children in *The Lancet* of 1844, and in 1861, at the Obstetrical Society of London, he read the much extended account of the disorder which appears elsewhere in this *Bulletin*, based on some 200 cases observed in his own practice.

Much of Little's early work was summarised in his "*Treatise on the Nature of Club Foot and Analogous Disorders*", published in 1839. He included histories of acquired as well as congenital cases in this book, but he apparently did not recognise that many of the acquired disorders were due to poliomyelitis. It remained for the German neurologist, Jacob von Heine, a year later, to differentiate acute anterior poliomyelitis from other forms of paralysis and to describe the deformities it might cause. Heine also drew attention to congenital spastic diplegia, though his description was not nearly so clear-cut and complete as that provided subsequently by Little. In 1843 Little wrote a monograph "*On Ankylosis or Stiff Joint*", and in 1853 his most substantial work appeared—"*On the Nature and Treatment of the Deformities of the Human Frame*"—containing one of the earliest descriptions of progressive muscular atrophy, in addition to accounts of the diseases particularly associated with Little's name.

Of modest and retiring disposition, Little is said to have had few recreations outside his professional work. After retiring from the active staff of the London Hospital in 1863, he continued his consulting practice in Brook Street and later in Park Street. In 1884 he finally retired to Ryarsh, near West Malling, in Kent, where he died on July 7, 1894, aged 84. He had four sons and seven daughters, and two of the sons became distinguished surgeons.

W. J. Bishop

ON THE INFLUENCE OF ABNORMAL PARTURITION,

DIFFICULT LABOURS, PREMATURE BIRTH, AND ASPHYXIA NEONATORUM, ON THE MENTAL AND PHYSICAL CONDITION OF THE CHILD, ESPECIALLY IN RELATION TO DEFORMITIES*

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(Communicated by DR. TYLER SMITH.)

PATHOLOGY has gradually taught that the fœtus in utero is subject to similar diseases to those which afflict the economy at later periods of existence. This is especially true if we turn to the study of the special class of abnormal conditions, which are termed deformities. We are acquainted, for example, with abundant instances of deformities arising *after* birth from disorders of the nervous system—disorders of nutrition, affecting the muscular and osseous structures—disorders from malposition and violence. Each of these classes of deformity has its representative amongst the deformities which originate before birth—viz., congenital club-foot, congenital rickets, congenital degenerations of muscles, amputations in utero from strangulation by umbilical cord or adventitious bands, intro-uterine fractures, etc.

There is, however, an epoch of existence—viz., the period of birth—during which, at first sight we might consider that the fœtal organism is subjected to conditions so different from those of its earlier and of its prospective later existence, that any untoward influences applied at this important juncture would affect the economy in a manner different to the influences at work during the periods ordinarily characterised as those of before birth and after birth.

The object of this communication is to show that the act of birth does occasionally imprint upon the nervous and muscular systems of the nascent infantile organism very serious and peculiar evils. When we investigate the evils in question, and their causative influences, we find that the same laws of pathology apply to diseases incidental to the act of birth as to those which originate before and after birth. We are, in fact, afforded another illustration that there exists no such thing as exceptional or special pathology.

Thirty-five years ago the pathology of deformities, if not invested with fable, was wrapped in obscurity; it was then scarcely perceived that the materials for extensive inductive observation existed.

Nearly twenty years ago, in a course of lectures published in the *Lancet*, and more fully in a *Treatise on Deformities*, published in 1853, I showed that premature

* *Trans. obstet. Soc. Lond.*, 1861-62, **3**, 293.

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birth, difficult labours, mechanical injuries during parturition to head and neck, where life had been saved, convulsions following the act of birth were apt to be succeeded by a determinate affection of the limbs of the child, which I designated spastic rigidity of the limbs of new-born children, spastic rigidity from asphyxia neonatorum, and assimilated it to the trismus nascentium and the universal spastic rigidity sometimes produced at later periods of existence.

Dugès, Cruveilhier, Smellie, Davis, Evory Kennedy, Doherty, Weber and Hecker, who have described the condition of stillborn children, suspended animation, asphyxia neonatorum and apoplexy of new-born children, are almost entirely silent respecting the after consequences to the infant, when not fatal. The first named is the only one who distinctly enunciates that hemiplegia and idiocy may follow injury received at birth. The others seem quite unaware that abnormal parturition, besides ending in death or recovery, not unfrequently has another termination—*i.e.*, in the language of medical writers, has a third termination "in other diseases." My friends, Drs. West, Tyler Smith, and Barnes, have informed me that instances of such a termination of abnormal labour have not fallen under their notice. Dr. Ramsbotham says he can remember two instances. It is obvious that the great majority of apparently stillborn infants, whose lives are saved by the attendant accoucheur, recover unharmed from that condition. I have, however, witnessed so many cases of deformity, mental and physical, traceable to causes operative at birth, that I consider the subject worthy the notice of the Obstetrical Society. In orthopædic practice alone, during about twenty years, I have met with probably two hundred cases of spastic rigidity from this cause. I omit reckoning the subjects of idiot and other asylums, in which probably such cases abound, but of which I have been able to obtain no history. I revert to the subject at the present moment because I believe I am now enabled to form an opinion of the nature of the anatomical lesions and the particular abnormal event at birth on which the symptoms depend. Moreover, as the study of the proximate cause of the affections which I shall describe requires the light of such facts as the members of this Society have peculiar opportunities of supplying, I make no further apology for occupying the Society's time

Before I describe the mental and physical derangements of the infant which can be referred to the effects of abnormal parturition and asphyxia at birth, I may be permitted to dwell upon the principal phenomena which occur in the fœtal organism immediately before, during, and immediately after the act of normal parturition.

The fœtus, during the long nine months of its abode in the uterus, has been justly regarded as being plunged into a deep sleep, giving no signs of existence as a semi-independent organism but by an occasional strictly reflex or convulsive movement of the limbs, and the pulsation of its heart during the later months of gestation. The materials for its nutrition, oxygenation of its blood, and the carrying off of the carbon, and probably of other residues of the metamorphosis of its tissues, being provided for by the intercourse which takes place between its blood and that of the mother at the placenta. With the commencement of normal parturition the long repose of the fœtus is broken in upon. The fœtus, during the uterine contractions, especially after evacuation of liquor amnii, is subjected, together with the placenta and umbilical cord, to a gradually increasing amount of pressure, through which it may reasonably be conjectured that the circulatory system, and consequently the capillary system, as of the lungs and nervous centres, are gradually prepared for the altered offices which are about to devolve upon them. This pressure is at first intermittent, the

duration of the period of repose at first greatly exceeding the period of disturbance; as the final exit approaches, the pressure simply remits, until at length it is so considerable that prompt escape from the mother alone prevents mischievous results to the nascent organism. During the uterine contractions a certain amount of impediment to placental respiration or to placental interchange of material is unavoidable, so much of undecarbonized or deteriorated blood is contained in the fœtal tissues—amongst other tissues, in those of the excitor of respiratory acts, the medulla oblongata—as suffices to give notice to the medulla oblongata of the need of inspiratory movements and of the admission of air into the lungs. Hence is explained the first-observed phenomenon of normal, independent, extra-uterine existence, the effectual act of inspiration, accompanied with the welcomed, characteristic, expressive cry of the new-born child. The normal impediment to placental interchange reaches its maximum at the moment of birth. Should any departure from the normal act of birth take place, should the act of normal respiration not be established at the moment of birth, the child presents itself in a state either of manifest death (stillborn), apparently stillborn, or in a state of more or less completely suspended animation, and does not utter the characteristic expressive cry of the new-born child.

The new-born child that has not yet attained to thorough independent existence tolerates a longer duration of suspended animation than the child in which pulmonary respiration has been thoroughly established or than the adult; yet reflection on the nature of a delay of only a few moments in the substitution of pulmonary for the ceased placental respiration would lead to the apprehension that even the want of a few breathings, if not fatal to the economy, may imprint a lasting injury upon it. The observations I have recorded of the direct connection between suspended animation at birth and mental and physical impairment of the individual, prove that the proportion of entire recoveries from the effects of asphyxia neonatorum is smaller than has hitherto been supposed.

It will be acknowledged that the state of things in the fœtus at the moment of birth, at the moment of entire withdrawal of placental or maternal circulatory influence, is one of imminent failure in decarbonization of the blood. If pulmonary respiration be not immediately established, the state of suspended animation—asphyxia neonatorum—takes place. From analogy with other forms of suffocation in later life, as from drowning, when the air-passages are suddenly and forcibly obstructed, suffocation also from inhalation of certain gases which exclude oxygen from the lungs, we may infer that the want of respiration in the new-born child is followed by stagnation of blood in all the large venous channels. We may direct our thoughts to the necessary consequences of blood stagnation in the sinuses of the brain, the venous plexuses surrounding the spinal cord, the venæ cavæ, the right ride of heart, and the pulmonary system. We can apprehend the inevitable congestions of the capillary system of the brain and spinal cord, and a prompt result in death, if the mischievous circle of affairs is not relieved by suitable respiration.

The forms of abnormal parturition which I have observed to precede certain mental and physical derangements of the infant consisted of difficult labours—*i.e.*, unnatural presentations, tedious labours from rigidity of maternal passages or apertures, instrumental labours, labours in which turning was had recourse to, breech presentations, premature labours, and cases in which the umbilical cord had been entangled around the infant's neck or had fallen down before the head. To these abnormal forms of labour I believe Cases LII and LIII justify me in adding labours in which, from want of due attention immediately after birth or after expulsion

from the mother, the child has been partially suffocated in the maternal secretions or under her clothes.

Doubtless in some of the instances I have recorded sufficient mechanical injury to head and neck was inflicted to account for whatever unfavourable consequences, whether these were fatal or not, may have ensued, but the more the facts I shall adduce are studied the more apparent, in my opinion, it will be that a larger proportion of infants, either dead, stillborn, apoplectic, or asphyxiated at birth, have been rendered so by interruption of the proper placental relation of the fœtus to the mother, and non-substitution of pulmonary respiration, that from direct mechanical injury to the brain and spinal cord.

Until quite recently the morbid anatomy of children dead at birth or shortly afterwards had been little recorded. Jadelot (*Traitê des Maladies des Enfants de Michael Underwood*, 1823, p. 67) says —"We never find effusion of blood, but only very considerable engorgement." Cruveilhier (*Anat. Pathol. sur l'Apoplexie des Nouveaux-nés*) and Dr. Evory Kennedy ("On Cerebral Apoplexy of New-born Infants," *Dublin Journal*, vol. x, p. 425) agree that effusion of blood takes place commonly on the surface or base of brain, never into the substance itself, and that in the majority even of fatal cases only intense turgescence of sinuses and veins, with extreme congestion of the capillary systems are found. Cruveilhier found in all cases the dura mater of spinal canal distended with fluid blood. The fullest contributions to the morbid anatomy of stillborn children have recently been made by C. Hecker, of Berlin, and F. Weber, of Kiel. Hecker (*Verhandlungen der Gesellschaft von Geburtskunde*, Berlin, 1853), after quoting Schmidt, Ritgen, Litzmann, and Krahmer, to the effect that children dying at birth or subsequently asphyxiated present numerous dotted, petechiæ-like ecchymoses on the surface of the lungs and diaphragm, gives a larger number of his own dissections, proving beyond a doubt that punctiform ecchymoses are present, as a rule, on the serous surfaces of chest and abdomen, sometimes on the skin, besides intense congestion of viscera of chest and abdomen, blood extravasations between pericranium and cranium, the vessels and sinuses of brain gorged with blood, in children born dead, whether from interruption of placental or insufficient pulmonary respiration, *caused by pressure on umbilical cord, premature separation of placenta, and uterine hæmorrhage*. Hecker also found several times *in prematurely born* children, who had lived a longer or shorter time after birth, *similar ecchymoses on surfaces of lungs and heart*.

F. Weber (*Beiträge zur Pathalogischen Anatomie der Neugebornen*, Kiel, 1851-54) found laceration of dura mater and effusion of blood between it and the bones, rupture of longitudinal and transverse sinuses of brain and considerable hæmorrhage on the surface and base of brain, sometimes sufficient to envelop cerebellum and oblongata in cases in which mechanical injury to bones of the head had occurred, whether or no instruments had been used to complete the delivery. But Weber found pretty generally the same tendency to punctated, capillary apoplexies, especially on the serous membranes of lungs, heart, brain, and spinal cord, as were first described by Hecker as a cause of death of new-born infants. The class of lesser injuries uniformly met with in death of child from abnormal labour was, according to Weber, great congestion of all the large veins and sinuses, intense congestion of surfaces of brain and spinal cord. In the spinal cord the small extravasations of blood and the congestion of pia mater were always greatest in the cervical and lumbar portions. Weber rationally accounts for the comparative infrequency of capillary apoplexy of the brain in these dissections, on the ground that capillary apoplexy is

usually recovered from, whilst extravasated blood oftener kills. The sequel will show that capillary apoplexies of the nervous centres are probably the cause of, at least, one form of persistent deformity of limbs—general spastic rigidity, and that although capillary apoplexy may not commonly destroy life, its consequences seriously impair the organism.

Weber describes an autopsy similar to some of those of Hecker, showing the results of great congestion, apoplexy of the pleura, congestion of brain, bloody serum in ventricles, the cerebellum and medulla oblongata swimming in it, in a case of death through descent of umbilical cord, *no pelvic obstruction having existed.*

The detailed autopsies of Hecker and Weber, with the carefully appended histories of the nature of the fatal impediment at birth, have greatly facilitated an explanation of the spastic rigidity and paralysis of limbs, which appeared from my observations to be produced by so many different forms of unnatural parturition. The dissections of these obstetricians show the important fact that mechanical injury of the fœtal head, neck, or trunk, is not necessary for the production of intense congestion and blood extravasation of serous surface of chest, brain, and spinal cord. The other phenomenon commonly observed in difficult and abnormal parturition is that of interruption of placental respiration and circulation with non-substitution of pulmonary breathing and circulation. To this phenomenon alone, when mechanical injury or impediment has not existed, can we attribute the internal congestions, capillary extravasations, serous effusions which correspond with or produce the symptoms of asphyxia, suspended animation, apoplexy, torpidity, tetanic spasms, convulsions of new-born children, and the spastic rigidity, paralysis, and idiocy subsequently witnessed. I am justified in regarding the dissections of Hecker and Weber as confimatory of the opinion emitted by me, that asphyxia neonatorum, through resulting injury to nervous centres, is the cause of the commonest contractions which originate at the moment of birth, namely, more or less general spastic rigidity, and sometimes of paralytic contraction.

The former class of affections may be described as impairment of *volition*, with *tonic* rigidity and ultimately structural shortening, in varying degrees, of a few or many of the muscles of the body. Both lower extremities are more or less generally involved. Sometimes the affection of one limb only is observed by the parent, but examination usually shows a smaller degree of affection in the limb supposed to be sound. The contraction in the hips, knees, and ankles, is often considerable. The flexors and adductors of thighs, the flexors of knees, and the gastrocnemii, preponderate. In most cases, after a time, owing to structural shortening of the muscles and of the articular ligaments, and perhaps to some change of form of articular surfaces, the thighs cannot be completely abducted or extended, the knees cannot be straightened, nor can the heels be properly applied to the ground. The upper extremities are sometimes held down by preponderating action of pectorals, teres major and teres minor, and latissimus dorsi; the elbows are semiflexed, the wrists partially flexed, pronated, and the fingers incapable of perfect voluntary direction. Sometimes the upper extremities appear unaffected with spasm or want of volition, sometimes a mere awkwardness in using them exists. Not infrequently the parent reports that the hands were formerly affected. Participation of the muscles of trunk is sometimes shown by the shortened, flattened aspect of pectoral and abdominal surfaces, as compared with the more elongated and rounded form of the back. The prominence of back partially disappears on recumbency, but the greater weakness of muscles on dorsal aspect of trunk is obvious when the individual again attempts to sit upright. The muscles feel harder than natural to the age. Micturi-

tion is sometimes observed to be rare, and the bowels usually confined either from deficient exercise of voluntary expulsive power or from implication of the sphincters. The muscles of speech are commonly involved, varying in degree from inability to utter correctly particular letters up to entire loss of articulating power. Sometimes articulation is only slow and difficult, like other acts of volition, the child or adult reminding us of a tardigrade animal. Sometimes the speech is nervous, impulsive, or stuttering. Often during the earliest months of life deglutition is impaired, and the power of carrying saliva into the fauces is not acquired until late. The intellectual functions are sometimes quite unaffected, but in the majority of cases the intellect suffers—from the slightest impairment which the parent unwillingly acknowledges or fails to perceive up to entire imbecility. The functions of organic life are unexceptionably performed, except, perhaps, that of development of caloric, although the depression of temperature in later life is more probably dependent upon the want of proper exercise. The frame is often lean and wiry, but not wasted. On the contrary, it is generally well nourished. The appetite is good, the child is often described as the healthiest of the family. These subjects often lead a more precarious existence during the first weeks after birth, at first even vegetative existence languishes, sometimes, perhaps, because premature birth or difficult labour, by impairing the maternal supply of nutriment, renders more difficult the infant's recovery from the shock its system received at birth. However, in the majority of instances, after restoration of the vegetative functions, a gradual but slow amelioration of all the functions of animal life is perceived. Some cases present distinct convulsive twitchings of face or limbs during first days after birth, open or suppressed convulsions, opisthotonos, or laryngismus. In some instances the persistent rigidity of muscles commences or is observed shortly after birth, in others it escapes observation until the lapse of some weeks or months. The child's limbs are sometimes reported to have been simply weaker, to have shared in the general debility, the question of viability having alone occupied the attention of the attendants during the first month. Occasionally the weakness of the limbs has been recognised as a genuine paralysis in the first instance, of which the rigidity of muscles has been the sequel. Before the age of three or four months, though sometimes in slight cases not until the ordinary time for walking arrives, the nurse perceives that the infant never thoroughly straightens the knees, that these cannot be properly depressed or separated, that she is unable to wash and dress the infant with the ordinary facility, that the hands are not properly used. The upper extremities recover before the lower limbs. Sometimes the trunk is habitually stiffened, so that the infant is turned over in the lap "all of a piece," as the nurse expresses it. Occasionally the head is habitually retracted. Where the symptom of convulsions or "inward convulsions" exists, the rigidity is attributed to the convulsions. In many cases convulsions have been absent. As the child approaches the period at which the first attempts at standing and progression should be made, it is observed to make no use of the limbs, or he is incapable of standing except on the toes, or the feet are disposed to cross each other. Even children slightly affected rarely "go alone" before three or four years of age, many are unable to raise themselves from the ground at that age, and others do not walk, even indifferently, at puberty. On examination, the surgeon finds that the soles of the feet are not properly applied to the ground, that the knees always incline inwardly, and continue bent. When locomotion is accomplished, the movements are characterised by inability to stand still and balance the body in erect attitude. In the best recoveries from *general* spastic rigidity, even in the adult,

the gait is shuffling, stiff; each knee, by forcible spastic rubbing against its fellow, obstructs progression.

The external form of the cranium occasionally exhibits departure from the normal or average type, such as general smallness of skull, depression of frontal or occipital region only, sometimes one lateral half of skull, sometimes of one half of occiput, or forehead only. In slight cases the head has been well developed.

In cases even with great inertia as to exercise of volition in any part of the body, common sensibility appears little, if at all, deficient. The child often, indeed, manifests uncommon sensitiveness to external impressions, even when approaching adolescence he is alarmed at trifling noises. The sleep after the first week of life is light, easily disturbed. Often there is extreme sensibility to touch, the whole condition reminding the observer of tetanus. In a few cases a distinct resemblance to severe chorea is perceptible. It is probable that some of the cases designated by authors congenital chorea have been cases of the affection I have described.

Amongst the more uncommon consequences of difficult or premature labour and asphyxia, I may refer to Cases XLVIII and XLIX, in which wry-neck apparently resulted from one or other of these causes, and Cases VII, X, XX, and others, in which a distinct hemiplegic contraction resulted. I have occasionally met with the slightest amount of single spastic talipes equinus referable to this cause. Such a case has commonly been attributed to dentition, a fit or illness during infancy, the first link in the pathological chain of nervous susceptibility caused by the asphyxia having been disregarded or overlooked.

A survey of the history of forty-seven cases, appended, shows that one fact is common to all the cases of persistent spastic rigidity of new-born children, namely, that some abnormal circumstance attended the act of parturition, or rather, the several processes concerned in separating the fœtus from the parent and its establishment in the world as an independent being. I cannot recall positively to mind, or find recorded in my journals, more than a single case in which this persistent *spastic* rigidity affected a numerous series of muscles of the trunk and extremities which could be unequivocally referred to any illness subsequent to the establishment of proper pulmonary respiration as its starting-point. Often it has been found that convulsions in infancy had occurred, to which the disease had been attributed. Spastic contraction of a single set of muscles, as the gastrocnemii of one or both limbs, commonly of one limb only, or of the muscles of the forearm and calf on one side, is certainly an everyday occurrence after infantile convulsions, convulsions *during* dentition, and during early childhood without convulsions or other marked illness. But general spastic rigidity I have, with one exception, found to have been preceded by some abnormal act connected with mode of birth. Occasionally several causes, either of which may be competent to produce cerebro-spinal disorder and deformity, may coexist. Thus, in Case XLIII uterine hæmorrhage occurred two months before labour; labour was tedious, accoucheur was absent at birth, the child was born with navel-string around neck and legs, and did not cry for an hour afterwards, and a large, hard substance, as large as another child—possibly a blasted twin conception —was discharged with the afterbirth. I may remark that asphyxia neonatorum, from whatever amount of disturbance in separation of fœtus from the uterus it may have resulted, is, as might be surmised, very apt to be accompanied with, and to be succeeded by, convulsions at variable periods after birth. It will be borne in mind that convulsions at birth, or subsequently to it, are but a symptom of lesion of nervous centres, and that we cannot refer one symptom of disorder of the nervous system to another symptom of the kind. The convulsions may doubtless react upon

the nervous centres, upon the lungs and heart, and probably aggravate the disorder. North (*Practical Observations on the Convulsions of Infants*, 1826, p. 52) says—"It cannot be doubted that convulsions occasionally arise from excessive and long-continued pressure of the head during protracted labour. . . . They generally pass off in a very short time after birth." But he adds, speaking of asphyxia, "If such an infant only partially recovers, and convulsions succeed, the death of the child almost inevitably follows." Cases II, IV, XVII, XXXIV and XLVI, in Appendix, are instances of recovery from such convulsions, and of production of subsequent spastic rigidity. North quotes Dr. Clark, to the effect that such children, if saved from immediate death, are liable to die suddenly in a fit of convulsions. I have witnessed several confirmations of this statement. Case III is an example of the kind. Baume (*Convulsions dans l'Enfance*, 1805, p. 69) makes a similar observation; and Smellie (*Midwifery*, 1772, vol. i, p. 230) alludes to convulsions before or soon after delivery from compression of head, to the danger, and oft-times the destruction, of the child. Billard (*Diseases of Children*, translation of third French edition, 1839, p. 472), speaking of the morbid anatomy of diseases of cerebro-spinal apparatus developed after birth, says—"The length of the labour, the necessary tractions in certain manœuvres, the difficulty with which respiration is established, the changes which the circulation undergoes, explain why the cerebro-spinal system is so often the seat of sanguineous congestions varying from simple injection of the meninges to true apoplexy." At p. 477 he remarks that in two-thirds of the cases of convulsions in new-born children, examined post mortem, spinal meningitis was found; myelitis was less frequently met with. Billard was not aware that cerebro-spinal congestions and apoplexy occur in infants where the labour had not been difficult, tedious, or involved mechanical aid—after descent of and pressure upon umbilical cord only, for example. The Appendix shows that Brachet (*Traité des Convulsions des Enfants*, 1837, p. 97) is too absolute in the statement that "the infants who are born after difficult and protracted labour are all, without exception, doomed to frequent convulsions."

It is impossible not to connect the persistent affection of the intellect, of volition, and of organic life, with the injury the several nervous centres suffered in some instances before the fœtus had reached the maternal pelvis, in others whilst in transit through it; and in a third set of cases, where the fœtus was exposed to neither of these kinds of injury, it suffered from asphyxia neonatorum, suspended animation, and its concomitant congestions, effusions, capillary apoplexies of brain, medulla oblongata, and spinal cord. Hitherto I have been afforded only one opportunity of learning the post-mortem condition of any of the cases of spastic rigidity which I have referred to asphyxia at birth, viz., Case LX, kindly furnished by my colleague, Dr. Down. It is certain that if examined after death, after living many years, and such cases I find may live at least past the meridian of life, an anatomical condition very different from that present at or soon after birth would be found. Without going so far as Weber, as to assert that capillary apoplexies are necessarily absorbed when immediate death does not result from them, we may conclude that although the effused blood-particles may be absorbed, permanent lesion—atrophy of the nervous tissue—results (see Case LX). Possibly a state of chronic meningitis, with effusion, or of chronic meningeal hyperæmia or congestion, or a certain amount of chronic myelitis, may maintain the spastic excitable tetanoid, sometimes choreal, contractions, with rigidity of the trunk and extremities. My experience as Physician to the London Hospital has afforded me some facts which support the idea that spinal meningitic and myelitic affections

may play a considerable part in the phenomena of spastic rigidity. Thus the only case of persistent general spastic rigidity of upper and lower extremities, commencing after adult age, which I had the opportunity of seeing at intervals during twenty years, and the general appearance of which appeared to me similar in many respects to spastic rigidity from asphyxia neonatorum, was found by me after death to have depended upon chronic spinal meningitis and myelitis. A case related by Cruveilhier, of pus found in medulla spinalis, in a case of death of infant on the fifth day after difficult labour supports this view.

The greater or smaller impairment of intellect may safely be attributed to the greater or less mischief inflicted upon the cerebrum. As already observed, the considerable extravasations of blood on the surface of the brain are usually fatal. The autopsy, Case LX, showing cicatrised apoplexies on surface and interior of brain, is an exception. The only fatal instance of partially stillborn infant, which I have had the opportunity of post-mortem examination, was one which came rapidly into the world, preceded by uterine hæmorrhage, nearly at full time, owing to fright to which the mother was exposed. Death of child ensued seventy hours after birth. In this case considerable effusion of blood was discovered in both ventricles of brain —a true apoplexy in the new-born child without mechanical injury. The autopsy, Case XLI, illustrates congestive apoplexy, no pelvic obstruction having existed.

I formerly found much difficulty in the analysis of various symptoms met with in different cases of spastic rigidity traceable to something abnormal in the act of birth. It soon became apparent that the symptoms, *of the living* at least, attributable to mechanical injury of head were a minority of the whole. This is consistent with the remark of Ollivier (*Traité sur les Maladies de la Moelle Epinière*, vol. i, p. 152), that whilst at natural birth the spinal cord is perfectly developed, the brain is still in a very rudimentary state, and consequently able to bear considerable disturbance without ultimate injury to its functions. In fact, in the new-born child brain-life is entirely absent: any injury it may have received at birth is at that period unaccompanied with special brain-symptoms, and, if not too severe, the child may entirely recover. Ollivier says (p. 244) "the brain of the new-born child is often found softened and destroyed without any external sign having permitted the practitioner to suspect it during life." In the present day, with the experience we now possess of the causes of death at or shortly after birth, the accoucheur will suspect the existence of some form of apoplexy in every case.

The severe lesions caused by mechanical compression and laceration, and extensive hæmorrhages within the skull, when they do not destroy life, give rise to permanent deformity of cranium, to atrophy of injured portions of brain, and are the cause of many cases erroneously described as *congenital* idiocy. Dr. J. Crichton Browne ("Psychical Diseases of Early Life," *Journal of Mental Science*, April 1860) is one of the few observers who have traced idiocy to difficult labours (see also Dr. Howe, *Causes of Idiocy*, Edinburgh, 1858). But in addition to the undoubted instances in which cranial injury and some imperfect development of intellect stand in the relation of cause and effect, the Appendix shows impaired intellect in Cases IV and VIII, in which no mechanical injury had taken place, but in which suspended animation, asphyxia neonatorum, and probably its consequent general and capillary congestion and ecchymoses —capillary apoplexies of the brain as well as of the spinal cord—perhaps even a moderate amount of larger apoplectic extravasation, had taken place, and had been imperfectly recovered from. I have observed that in impaired intellect from abnormal birth the degree of impairment met with in private practice often does not exceed feebleness of intellect; it varies

much in degree, as elsewhere mentioned; it is often not sufficient to exclude the individual from family society. The individual may acquire a fair knowledge of music, the memory is good, the constructive tendency may exist, a fair capacity for arithmetic and languages may be displayed, but there commonly exists a great want of application, a slowness of intellect similar to the slowness of volition. In other cases, where intellectual powers are good, a preternatural impulsive nervous condition of mind exists, combined with an agitated, eager, anxious mode of performing acts of volition. Making every allowance for family peculiarities, there undoubtedly exists a considerable pathological resemblence, even in intellectual character and physiognomical expression, in these subjects of more or less general spastic rigidity. The occurrence of this feeble intellect in those who have not been exposed to mechanical injury of head, but in whom premature birth or pressure on umbilical cord has been recorded, appears explicable only on the supposition that the asphyxia and feebleness at birth had been followed by the usual capillary or larger hæmorrhage or effusions in brain, and their transformations and consequences to the nervous tissue; and the degree and variety of impaired function of brain may be due to the degree and variety of situation of these hæmorrhages.

The affections of the functions of organic life, the protracted inability to suck and swallow in a natural way, often observed during the first few weeks of life, the liability to "choking noises in the throat" and other signs of what may be classed under the name laryngismus stridulus, and the affections of speech dependent upon impaired innervation of glottis, pharynx, tongue, and lips, and consequent arrested development of some of these parts (larynx), may be referred to injury at base of brain and medulla oblongata. An occasional choke and gasp for breath, succeeded by a sigh, was described by the nurse of Miss N. (Case XXX) as having continued to the date of the report, when the patient was twelve years old. Occasionally the injury to base of brain or medulla oblongata may have resulted from mechanical displacement of part of the occipital bone, as in some cases described by Dr. J. Marion Sims (*American Journal of Medical Science*, April 1846) under the title of Tetanus of New-born Children; in Case LV of my own in Appendix the injury was consequent upon violent traction exercised to extricate head in breech presentation. But as in the explanation I have given of the causes of impaired intellect in relation to supposed injury to brain at birth, so I can show by reference to Cases IV, VI, XI, XII, and others, in Appendix, that mechanical injury to base of cranium and neck in those who survive is only exceptionally the cause of difficult deglutition, respiration, and speech, but that these important symptoms occur in practice oftenest in cases in which suspended animation or asphyxia at birth took place without previous violence to head and neck, rendering it probable that capillary apoplexy, serous or sanguineous effusion towards base of brain and in and around medulla oblongata, resulted from the general blood-stasis accompanying the asphyxia. Joerg (*Kinderkrankheiten*, p. 387) says that immediate death, although the heart continues to pulsate for several minutes after birth, follows mechanical injury, such as stretching or twisting, of cervical vertebræ. Case LV, with which I was favoured by Dr. M'Intyre, of Odiham, and probably Cases XLVIII, by Mr. Brown, of Camberwell, and XLIX, show that children recover from the immediate consequences of considerable injury in this situation. Dr. Marion Sims (opus cit.) describes, under the head of trismus nascentium, well-marked instances of spastic rigidity of new-born children. One case was that of a negro, a twin, the second born; labour was tedious, the child stillborn, several minutes having

elapsed before respiration was established. Tetanic symptoms were discovered on the sixth day, succeeded by death in ninety-six hours. At the autopsy "coagulum of blood was found occupying the whole length of spine, perfectly enveloping the medulla spinalis, thicker as it approached the brain. Spinal veins full of black blood." Dr. Sims attaches no importance to the tedious labour nor to the asphyxia at birth. Of six cases of trismus of new-born children reported by him, incidental mention is made of two of them having ensued after difficult labours. In some later cases of trismus nascentium published by Dr. Sims (opus cit., 1884), either inability to suck or stridulous breathing were observed soon after birth. It appears probable that the usually fatal disorder denominated trismus nascentium is often induced by the same causes—asphyxia at birth—and when recovered from has constituted the early stage of the condition which I have so often met with in older children, and have denominated spastic rigidity from asphyxia at birth. Abercrombie (*Diseases of Children*, sect. iv, Case 150) describes a case of spinal apoplexy of an infant who has been unable to suck and died with trismus and convulsions on the eleventh day. At Case 147 he speaks of hematorachis causing tetanus of new-born child. Weber (opus cit.), in death from trismus nascentium, always found the principal morbid appearances in spinal cord. Dr. Evory Kennedy ("On Cerebral Apoplexy of New-born Infants", *Dublin Journal*, vol. x, p. 429) relates a case of an infant which, after protracted birth and difficulty in establishment of its respiration, was seized on the second day after birth with general convulsions, hands clenched, screaming, abdominal muscles tense, respiration diaphragmatic, death on third day. At the autopsy, the vessels on hemispheres were much loaded, serous fluid abounded in spinal canal. The veins and membranes of medulla oblongata were excessively turgid and congested.

Among some interesting cases reported by Dr. Doherty (*Dublin Journal*, vol. xxv) of asphyxia of new-born children (a title to which he objects because the individuals have never breathed), are several which I recognise as belonging to the more numerous class of recovered asphyxia cases which present themselves in later life. Thus Dr. Doherty's Case 19, asphyxia of two hours' duration, resulted from prolapse of funis. Fifteen hours after birth convulsions set in; death on the fifth day. At autopsy—infiltration of blood into cellular tissue about dura mater of the cord, dura mater congested, vessels in spinal canal gorged, serous effusion in theca; sinuses of brain distended. *Blood between dura mater and parietal bones.* Dr. Doherty relates another case (Case 4) in which labour lasted three hours, funis expelled before head. Asphyxia reported, "followed by general tendency to spasm," said to have gradually recovered. The case, however, reappears as Case 28 a year afterwards, the child "never having been able since birth to hold up head." Dr. Doherty, however, doubts whether the symptoms were connected with the original transient apoplexy, as he properly designates the primary state. It was doubtless a case of asphyxia from descent of cord before head, accompanied with capillary or more extensive apoplexy or other effusion in nervous centres, followed by debility, paralysis, and spastic rigidity, similar to several of the cases I have appended. (Cases XIX, XLIII, XLV.)

Brachet, the author of the most complete work on the convulsions of children, relates a case of what he designates hereditary convulsions, overlooking the fact, which he incidentally mentions, that the child was semi-asphyxiated for half an hour after birth. He adds (p. 102), "I confess I could discover no exciting cause of the convulsions unless it were that M. Montain, the accoucheur, had been obliged to give the child some slaps on the buttocks to recall it to life." He adds,

"Quelque peu de confiance que j'ajoute à ce cause, elle est la seule probable, surtout chez un enfant qui y était disposé par sa constitution." Brachet also relates (p. 106) the case of a female child coming into the world after the mother had suffered two or three frights. The child's weakness was so great that the child did not cry for a fortnight, and swallowed with difficulty. This was succeeded by "convulsions neophytes de sauvages." She recovered, but he says, "pour la moindre cause elle tressaille et paraît menacée de convulsions."

It will be remembered that early in this paper I described this great susceptibility to impressions, almost tetanic, as a common accompaniment of spastic rigidity from asphyxia neonatorum.

Reference to more than fifty cases of injury of mind or body from abnormal parturition which are appended, will show that whilst in many cases the subsequent symptoms indicated that the brain and medulla oblongata had permanently suffered, the only one of the nervous centres which invariably presented symptoms of lesion was the medulla spinalis.

If—from analogy with the contractions of limbs observed to follow well-known diseases of spinal cord in later life, and from the fact of capillary apoplexy, larger blood-extravasations, and serous effusions being met with after death in spinal cord of infants who have died stillborn from premature birth, descent of funis before head, etc., without mechanical injury to head and neck—I am justified in referring the spastic rigidity which follows asphyxia at birth to lesion of spinal cord, and not to lesion of brain or medulla oblongata, it is obvious, from the greater frequency of this evidence of lesion of spinal cord than of lesion of brain and medulla oblongata, that from some cause this nervous centre suffers most often from the asphyxia, or least frequently recovers its integrity. It seems almost superfluous to add, as a further proof of non-dependence of spastic rigidity of limbs upon mechanical injury at birth, that the lower extremities are oftenest affected and are the slowest to recover, although they derive their nerve-power from the lower part of the spinal column, which is assuredly the part of the cerebro-spinal axis least obnoxious to mechanical injury.

When we consider the intimate pathological connection between spasm and paralysis it is remarkable that these cases of spastic rigidity from asphyxia at birth do not offer a decided combination of spasm and paralysis, such as is observed after ordinary cerebro-spinal disease in childhood. It is common, after such diseases, to find a child with one limb affected with paralysis or paralytic contraction and the opposite limb with spasmodic contraction.

As additional evidence of the dependence of the several states of brain, medulla oblongata, and medulla spinalis upon the asphyxia which so often attends abnormal parturition, I may recall to mind that recovery from asphyxia from chokedamp, asphyxia from suspension, are apt to be followed by cerebro-spinal disease; and I may add that at several autopsies after the asphyxia of Asiatic cholera, I have witnessed small blood-extravasations on serous surfaces of lungs and heart. Experiments on submersion of animals show internal congestion and ecchymoses of serous surfaces as a consequence of that form of suffocation.

Joerg (*Kinderkrankheiten*, 1828, pp. 402—438) is the only author I have met with who distinctly enunciates that too early and unripe-born fœtuses present a state of weakness, persisting in the muscles until puberty or later. He says it interrupts use of muscles during first and second periods of life, as well in limbs as in carriage of head and trunk, often thereby causing curvatures of spine and legs. Ollivier was aware of the liability of the spinal cord to suffer after difficult labour, for he says (opus cit., p. 240), "The greater influence of the spinal cord at birth

appears to continue during the first portion of extra-uterine existence, for affections of the spinal cord and its membranes sont assez communs dans les enfants *naissans*." Ollivier also distinctly attributes the marked injection of membranes of spinal cord in new-born children examined by him to the embarrassment which respiration and circulation undergo at this period of life.

It will be observed that I have in this paper often employed the term asphyxia neonatorum nosologically, in its widest sense, embracing in it all the conditions of suspended animation in the new-born infant which have for their result to prevent the immediate establishment of proper respiration and circulation, whether or no the colour of the infant be pale or dark. It is probable, from analogy with the asphyxias of later life, that the dark colour of the surface is a measure of the embarrassment of the pulmonic and cardiac functions, the pallor indicating greater prostration and greater tendency to cessation of nerve-life and death. D. D. Davies (*Principles and Practice of Obstetric Medicine*, vol. ii, p. 1212) endeavours in vain to establish a difference in the aspect of the infant, according to whether the state of suspended animation arises from asphyxia, asthenia, or apoplexy. C. A. Struve (*On Physical Education of Children*, translated by Willich, 1800) makes two kinds of apparently still-born, the adynamic and apoplectic. Joerg (opus cit., p. 402) recognises that the suspended animation is sometimes composed of two states, injury (mechanical) to head and want of air. It is apparent that the phenomena of suspended animation of infants after birth will permit of more extended observation by the members of the Obstetrical Society. Davis's view, that three states occur, asphyxia, asthenia, and apoplexy, is doubtless correct. It is, however, evident from that which I have already stated, that it is not yet possible before death to point out upon which of these three conditions the suspended animation depends. The want of breathing is manifest in all cases, asthenia may be present as a complication, and if the suspended animation terminates in death or in cerebro-spinal disorder, we may infer the existence of sanguineous congestions and apoplexies in the nervous centres.

This is the class of cases which, during the "sensation" times of the promulgation of subcutaneous tenotomy, furnished opportunity to an able French orthopædic surgeon triumphantly to divide sixty or more muscles at one sitting. Happily, Stromeyer's operation of subcutaneous tenotomy rested upon a more secure foundation than could be over-thrown by so great an abuse of it.

* * *

I trust the views of the pathology of the lesions of mind and body referable to the influence of the act of birth upon the child, which I hope to have somewhat unravelled, will promote the beneficial treatment of the disorders when detected in the early stages. In the later stages, the general principles of orthopædy, and mental training when the intellect is affected, are successfully applicable in the inverse proportion to the extent of the permanent disorganization of the nervous centres and of peripheral structures. The length to which this paper has already extended prevents my dwelling upon the subject of treatment. I have had many of these cases under observation from one to twenty years, and may mention as an encouragement to other practitioners that treatment based upon physiology and rational therapeutics effects an amelioration surprising to those who have not watched such cases. Many of the most helpless have been restored to considerable activity and enjoyment of life. Even cases which exhibit impaired intellect may be benefited in mind and body to an unexpected extent.

When we reflect on the frequency of pulmonary engorgement and ecchymosis as well in the interior of the lungs as upon their surface, also the distension of

venæ cavæ, the right side of heart, and the ecchymoses on pericardium, in the bodies of still-born children, it will appear not improbable that since one of the members of the tripod of life—the cerebro-spinal system—manifests defects in after-life referable to injury received at birth or to asphyxia neonatorum, in like manner partially still-born infants who recover with atelectasia pulmonum or with strained and injured hearts, may in after-life present anomalous affections of, or be prone to, pulmonary or cardiac disorder.

I would therefore suggest for inquiry, whether, for example, some cases of "congenital" cyanosis may not be induced at birth through impediment at this period to the normal substitution of infantile for the fœtal circulatory route, causing, for example, interruption of development and non-closure of foramen ovale. I am indebted to Mr. Curling for the following case. A youth, æt. 12 years, the second child born of parents not liable to asthmatic or other pulmonary complaints, did not cry immediately at birth, but received several vigorous slaps from the hand of the accoucheur before respiration was established. The child has from an early period of infancy been subject to considerable difficulty of breathing, and to attacks of acute dyspnœa on slightest cause. His asthmatic condition has puzzled several distinguished physicians who have seen him. Refuge has been taken in "*congenital* asthma." May not the starting point of the complaint have been injury to the capillary system or larger vessels of heart and lungs at the moment of birth?

The researches of Weber and Hecker into the morbid anatomy of still-born children testify also that the vascular system of the abdominal viscera undergoes disturbance, causing ecchymosis in those organs from the accidents attending birth.

It is further suggested, therefore, whether some of the ailments in these viscera, occasionally presenting themselves in the earliest periods of life, may not be due to causes similar to those which I have shown undoubtedly influence the cerebro-spinal system and its dependent organs. And lastly, as the general capillary system cannot be independent of that which affects the circulation of the brain, chest and abdomen, there remains for consideration whether the nutrition and development of the muscles and peripheral nerves are not directly affected, independently of the influence of the nervous centres upon them, by the proved abnormal congestions sometimes accompanying the act of birth.

APPENDIX OF CASES

The first column in Appendix of Cases contains the Number by which the case is alluded to in this communication. The second column contains the initials or name of the case, sometimes the date and name of physician or surgeon with whom the case was seen in consultation, and the number under which the case is recorded in my journal of similar and allied cases. The third column records the age in years when I first saw the case. The fourth column contains a literal transcript of the description of the case as entered in my journal at the time the case was first seen. The fifth column contains the history, mainly in the words of the informant. It is, of course, impossible to vouch for the accuracy of informant as to child being six months' child, etc., to a week or two.

Occasionally the report is brought by the author to the present time. This has not generally been done, in order to save space. The complete progress of the case was not required to be shown when treating of pathogeny.

Fig. 1.—Contraction of adductors and flexors of lower extremities. Left hand weak. Both hands awkward. More paralytic than spastic. Born with navel-string around neck. Asphyxia neonatorum one hour. See Case XLIII.

Fig. 2.—General spastic contraction of the lower extremities. Premature birth. Asphyxia neonatorum of thirty-six hours' duration. Hands unaffected. See Case XLVII.

Abstract of Cases of Spastic Rigidity. Labour Abnormal or Premature, or Asphyxia at Birth.

Case No.	*Initials and Date*	*Age (Yr.)*	*Description when First Seen*	*History obtained from Parents, etc.*
I	Esther T. (78.)	4	Contraction of flexors and adductors of lower extremities slight; left particularly affected.	Mother confined at seventh month of gestation through over-fatigue. Strabismus convergens of left eye. (A later child subject to fits; another never rallied at birth.)
II	Frances Ann F. 1840 (80.)	7	Spastic rigidity of adductors and flexors of thighs, flexors of knees, and posterior muscles of legs; left most affected.	Mother's first pregnancy; at full period; labour tedious and difficult, but "straight". Mother suffered from fright 2 months before confinement; never felt movements of fœtus afterwards. Child stuporous, or alternately crying or convulsed, 4-5 days after birth. Unable to suck until 5 days old. Ran alone on toes at 4 years. Intellect unaffected. "Irritable nervous system." General appearance of irregular muscular action.
III	Lydia C. April 1844 (81.)	6	Spastic contraction of lower extremities; knees separable 1 foot. Gastrocnemii. especially left, much contracted. Left arm contracted, but extensible. Makes slight attempts to walk.	Mother had great anxiety during gestation; a fright 2 days before confinement. Child insensible 2 or 3 days after birth. "Fits" after birth. Strabismus. Constipated. Says "mamma" and "papa" only. Expresses aversion and pleasure. Idiotic. Died suddenly in convulsions a month after report. Had previous convulsions occasionally, apparently excited by loaded stomach.
IV	Jane B. April 1845 (136.)	9	Spastic rigidity of gastrocnemii; slight of kness. Walking commenced at 6 years of age. Never balanced herself unaided, until use of high-heeled boots two years ago. Right arm inefficient through inability properly to supinate and extend it.	This was mother's third pregnancy. Suffered in health and spirits through death of a friend in her confinement. J. B. was born at eighth month; was "the smallest infant" seen. Apparently born perfect, but slept unusually much for the first fortnight. In convulsions from beginning of third week to twelfth week; never free for 5 minutes during that period, *i.e.*, not without "a hand, finger or toe being convulsed". Strabismus. Speech difficult. Intellect feeble; memory good. Docile, affectionate. jealous. In 1846, a year after first report, "has had governess and rubber constantly employed in her mental and physical education. Is much improved in talking and walking, and use of hands; begins the piano; more cheerful; anxious to improve".
V	Emma S. In consultation with Mr. Searle. 1845 (149.)	3¾	Slight rigidity of thighs, knees, and gastrocnemii. Thighs cannot be thoroughly separated. knees can with difficulty be straightened and the feet flexed only when the knees are bent. Right gastrocnemius more contracted. In 1850 reported equally contracted, left more deformed, owing to implication of foot adductors. "Hands less perfect than brothers and sisters."	Seventh month child. Birth-weight 2 lb. 8 oz.; cried, and took breast directly. Intellect good. "Said to be precocious." Frontal region narrow; excitable, passionate. In 1850, reported to walk with difficulty, timid, afraid of falling; has grown stout, excellent health, very active. Formerly never accustomed to sleep well; at present sleep is good. When standing, principal defect is inversion of left foot.

Case No.	*Initials and Date*	*Age (Yr.)*	*Description when First Seen*	*History obtained from Parents, etc.*
VI	Hannah D. August 1845 Seen with Mr. Kisch (159.)	11	Spastic contraction of lower extremities flexors, especially of gastrocnemii, and adductors. Occasional strabismus; left slightly worse. Slight projection of sternum.	First child. Born 3 or 4 weeks before time. "Mother was a week in labour." Infant emaciated. Did not cry for 3 weeks, and then not as another child; "couldn't breathe"; suppressed cry (laryngismus); deglutition was difficult, and remains slightly so. Never had a fit when awake, but ever since birth the slightest noise, or other disturbance during sleep, would excite starting and stiffening of limbs. At age of 9 or 10 months began to improve. Was weaned at 7 months. Showed signs of walking at 3 years old; always was weak in sitting up. Is round-shouldered, but no actual deformity of spine. Since age of 5 has walked with aid of hand on table, etc. Limbs always cold. Walks on toes unless reminded. Hands small; uses them perfectly. Everything done with hands and feet done with nervous quickness. Speech quick. The face looks inclined to be idiotic, yet intellect appears good. In 1848 reported much improved, morally, and physically; rarely raises heels; cannot walk quite unaided; starts like a narcotized frog when unexpectedly touched. Plays piano well. Subsequently went to reside in a hot climate, and reported to be much better in consequence.
VII	Charles W. 1845 (170.)	7	Spastic contraction of right arm and leg, flexors, adductors, pronators.	Labour lasted 24 hours, without instruments. Artificial respiration required for a quarter of an hour after birth. When a month old spastic and clonic movements observed. "Occasionally has a fit, which appears to affect throat only." Is never insensible in the fit. Habitual constipation. In 1847 "reads better".
VIII	Elizabeth W. 1845 (188.)	4½	Spastic rigidity of lower limbs, flexors and adductors, not extreme. Slight "weakness" of hands.	First child, at 7 months' gestation. Observed in earliest infancy to be stiff in the limbs. Cannot walk alone; at 3, moved with help of chairs. Crosses one leg over the other. Looks slightly imbecile. Noticed in 1855 that each patella, instead of occupying front of knee-joint, *i.e.*, situated within or upon the trochlea of the femur, is drawn up in front of femur above the trochlea. This makes it probable that this, which I have called spastic contraction of flexors and adductors, in reality affects all the muscles, extensors as well as flexors, the flexors in some parts merely preponderating.
IX	M. J. Male (199.)	3¾	Spastic contraction of lower extremities, flexors, and adductors. Slight contraction of upper extremities formerly observed.	"Child came unexpected, at 8 months." A brother, aged 7½ years, has slight contraction of muscles of planta, and the posterior leg muscles, left side. This originated at 15 months, after 2 or 3 days' febrile indigestion.
X	George A. H. N. 1848 (265.)	3½	Spastic contraction, flexors and pronators of right arm and leg. Backward in speech.	Born at full period: underwent turning; did not rally for half an hour after birth. Seen as lately as 1861. Appears to manage in society; not brilliant; obstinate. Walks like an adult who has rigid contraction from hemiplegia. Right arm still amost useless.

Case No.	*Initials and Date*	*Age (Yr.)*	*Description when First Seen*	*History obtained from Parents, etc.*
XI	M. S. Male 1849 (310.)	3	Contraction of gastrocnemii both sides, feet inclined to valgus. Doubtful whether spasm of muscles of calf, or paralysis, has existed; "probably only the result of the general muscular weakness observable in prematurely-born children."	Born at eighth month. Weak since birth; night screams; croupal cough and breathing. Takes less notice than usual at his age; laughs. Head inclined to heat! Hands have been weak; now used properly. Did not sit up until 18 months old. Bowels confined.
XII	M. M. Male 1850 Dr. Elliott, Dublin. (340.)	14	Spastic contraction of both lower extremities; right most affected. Adductors of thighs contracted, so that knees separate 7½ inches when thighs are extended, and 11 inches when thighs are flexed.	"Born at seventh month." At 2½ years walked with assistance. Deformity rapidly increased since age of 9 or 10 years. Slight difficulty of utterance when nervous. Intellect good.
XIII	Charles P. W. (392.)	11	Inordinate action of flexors and adductors of lower extremities.	First conception. A twin, the first born; 7½ months of gestation; labour protracted.
XIV	Patrick A. (400.)	6½	Spastic contraction of hips very slight. Spastic contraction of knees slight. Spastic contraction of feet considerable.	Seven months' child. "No asphyxia mentioned."
XV	Anastasia R. (405.)	10½	Spastic inability thoroughly to bend ankles.	Third child; born at 7 months; rallied half an hour after birth. Began to walk at 2 years. Was always weakly. Walked alone at 3½ years; shaky; gait always peculiar, liable to trip and fall. Intellect good, although head is small.
XVI	Henry S. 1852, in consultation with Mr. Brown, Stourport. (406.)	10	General spastic rigidity of lower limbs. Could never separate thighs.	Second child; seventh month of gestation; doubted for several days whether he would live; slept the first 10 weeks, during which he did not appear to grow. Always weak. Began to walk at 4 years old. Had formerly peculiar way of throwing head back. Bowels very confined. Micturition difficult. Left testis not descended.
XVII	A Male 1851, with Mr. Mallam, Hooknorton, and in 1861, with Mr. W. Adams.	6	General spastic rigidity, especially of right half of the body.	First child. Asphyxiated half an hour after birth. The pressure, not instrumental, over the occiput so great as to cause sloughing over occipital protuberance. Did not suck first 14 days; very feeble; unable to cry aloud; convulsions the first 48 hours. Dentition easy. Was a large child. Always unable to retain seat on knee of nurse. Twitchings of the limbs—choreal (?)—often observed, and left angle of mouth drawn down. At 5 years walked round the table, using it as a support. Report, 1861: Has grown tall and stout. More structural shortening. Increase of spasmodic jerkings. Less able to walk unsupported. Intellect impaired; cheerful; speaks

Case No.	*Initials and Date*	*Age (Yr.)*	*Description when First Seen*	*History obtained from Parents, etc.*
				distinctly, when he speaks slowly and is not agitated. (When aged 13 years, measures from ear to ear posteriorly, 6½ in.; over vertex 11½ in.; in front 10⅛ in. Left hemisphere feels smaller. Distinct depression felt in right occipital region. Circumference of skull at eyebrows, 20½ in.)
XVIII	John T. (455.)	24	General spastic contraction. Right less affected.	Eight (?) months' child. Had no nails at birth; lay as if dead 6 weeks; uttered no cry until that time. Strabismus convergens, more marked in right eye. Strabismus in the family. Always remarkably confined in bowels. Remembers reading before he walked.
XIX	Frederick J. (457.)	16	Spastic contraction of both lower extremities and right arm. Left arm weak only, and left leg was originally less affected.	At birth umbilical cord entangled neck; labour sudden; no signs of animation for 10 minutes. Speech difficult: x, b, m, worst letters. Observed never to sit up well.
XX	Miss R. (463.)	5	Spastic contraction of left arm and leg.	Foot presentation: legs and lower half of body born half an hour before head. Insensible 2 hours after birth. Is timid, nervous, falls with the slightest touch.
XXI	Henry W. Dr. Gall, 1854. (484.)	5½	Severe spastic contraction of left arm and leg.	Third child. Mother confined without any attendant; not seen to for many minutes; born 3 weeks before time; mother often had flooding before labour. Was very stout child Unable to walk or sit safely on floor. Looks hearty and intelligent. Until recently extremely constipated. Can only say "pony", "beer", and a few other words, imperfectly. Good letters, a, b, d, e, g, h, i, j, k, o, p, r, t = 13; imperfect, s, u; absent, c, f, l, m, n, q, v, w, x, y, z. (Convulsed in 1855 for 4 hours. Relieved by action of the bowels.)
XXII	Albert F. (485.)	3½	General spasmo-paralytic contraction: left side least affected.	Small child born; nearly dead; remained in convulsions 2 or 3 days. Can neither stand, feed himself, nor speak; looks imbecile, though said to be intelligent; very obstinate.
XXIII	Clement F. (567.)	6	Spasmo-paralysis. Right most affected.	Eight months' child. Child born with many bladders on back and stomach. Great weakness in neck. Cried 5 minutes after birth. Eyes opened 12 hours after. Mother, much reduced by illness, scarcely sensible at the labour. Infant extremely small and delicate. Slept first month; took breast at end of first week. Speech hesitating and indistinct; extremely shortsighted and nervous; easily startled. 1861: Very much improved.
XXIV	Gertrude P. (573.)	7¾	Spastic contraction of lower extremities Hands awkward only.	Seven months' child. Asphyxia some minutes; feeble a long time. Slight risus sardonicus; speech good, although lips look stiff. Is very "irritable".
XXV	Alfred L. (576.)	7	Spastic contraction of right side.	Mother in labour 24 hours; instrumental. Child black, supposed to be dead; restored

Case No.	*Initials and Date*	*Age (Yr.)*	*Description when First Seen*	*History obtained from Parents, etc.*
				with difficulty. Did not cry for half an hour. Head out of shape; long and high. Sudden noises uncommonly startle; is timid; can stand still, but has chorea-like movements.
XXVI	Miss N. (579.)	5	Slight spasmo-paralysis of right side.	Twin child, half the size of male child; was supposed to be dead; recovered under artificial respiration.
XXVII	James B. (581.)	12	Slight spastic contraction of lower extremities. The peculiar lameness occurring in prematurely-born children slightly evident.	Born between 8 and 9 months. Mother believes child cried immediately. Is cousin of 567. Slight strabismus.
XXVIII	Alex L. (582.)	5	Spastic contraction of lower extremities.	Seven months' child. Was reported as dead. Intellect very good; speech ditto.
XXIX	Lucia H. (584.)	14 m.	General spasmo-paralysis. Uses left arm best.	Born at full period. Asphyxia 2 hours.
XXX	Miss N. (584.)	6½	Severe spasmo-paralytic contraction of lower extremities.	Eight months' child, twin. Was born dark from bad circulation; expected during first night to die every minute; did not cry when born. Intellect good. Averse to lying on back, explained by tendency of body to roll on one side. Always confined in bowels; undue tendency to hold water.
XXXI	Emily T. (586.)	7½	Spasmo-paralytic contraction of left side.	Unusually large head; detained at brim and pelvis 4 hours; after pressing on perinæum 7 or 8 hours more elapsed. Partial asphyxia; gasping breathing 2 hours. Not dressed for 5 hours; cried vigorously at end of 3.
XXXII	Julie T. (591.)	13	Severe general spastic contraction, including wry-neck.	Labour difficult. No asphyxia. Peculiar spasm of limbs, and falling of head to one side, observed directly after birth.
XXXIII	Amelia S. Dr. Elliot, Stratford. (604.)	6½	Spastic contraction of lower extremities. Hands awkward.	Very small child, born before time. No asphyxia mentioned. Had screaming fits at 4 weeks old. Always confined in bowels especially first month.
XXXIV	Henry A. In consultation with Dr. Roberts, Salisbury, 1859. (606.)	4	General contraction of of lower extremities.	Bad labour: 24 hours; forceps applied. Child asphyxiated 2 hours; was black, swollen, born with wound on left forehead. Did not cry for 2 hours. Would not have been known to be alive but by gentle movements of the chest. Was cold all the time, although hot flannels were constantly applied. On coming to at the end of 2 days, had a convulsive fit, a "twittering" of the cheeks, thumbs and hands slightly clenched. These fits lasted, off and on, quite 3 weeks. Had no idea of sucking for 6 weeks. At first a single drop of milk put into the mouth caused great difficulty of swallowing. At present swallows more slowly than other children. Until a year ago would look frightened, pale, seem to have struggle in throat for an instant, then laugh as if relieved from some suffering. Intellect feeble, but actively notices objects. Tenotomy was performed a year ago. Bowels did not come right

Case No.	*Initials and Date*	*Age (Yr.)*	*Description when First Seen*	*History obtained from Parents, etc.*
				for 2 months. Liver extremely torpid. Head now somewhat conical in shape. At 6 months had every night, once or oftener, a fearful peculiar scream. At 9 months old had convulsive fit of 36 hours' duration. September 1861: reported to have walked alone within last 2 months. Intelligence much improved.
XXXV	Dora H. In consultation with Mr. Adams, Harrington Square, N.W. (619.)	3½	Spastic contraction of lower extremities; confined to calves.	Seven months' child. Lost blood from navel string. Moaned at birth. Expected not to live the first three days. Spent most time asleep. Limbs always crossed. Structural shortening of tendons and muscles observed when 8 months old. Strabismus observed during teething. No convulsions.
XXXVI	William J. F. In consultation with Mr. Ballard, 1860. (629.)	14	General spastic contraction of upper and lower extremities severe.	First child, full time, "cross birth"; was turned and brought down by feet. Labour lasted 36 to 40 hours; thought to have been injured at birth; did not cry for 2 hours; very dark; "lay for hours without taking notice"; unable to suck for a week. Mother noticed nothing else the matter, except that his head leaned to the left. Never would lie on floor, as if frightened; can crawl; has never walked. Still has difficulty in swallowing; a "choking". Left side of head smaller. Maxillæ large, especially upper. Nothing wrong with sphincters, except that he wets himself when excited. Weak intellect; can read an easy book. Speech tolerably distinct. Can write badly. Is moveable like a stiff skeleton. Sits badly, round-shouldered. Head inclines to left. (Father died of apoplexy. Family history otherwise good. Five brothers and sisters healthy.)
XXXVII	George H. Dr. Sturges, Sydney Sq. (652)	4¼	Spastic rigidity of lower extremities with structural shortening of gastrocnemii. Can just move one leg before the other; right least affected; elevation of heel, and disposition to walk on upper part of metatarsus and toes (equinus extreme).	First child. Said to have been born two months before time. Nails were perfect. Cried lustily at birth for 10 minutes. Unable to suck for 6 weeks. Sphincters always good. Considerable hollow in each infra-mammary region, more remarked owing to developed form of triangle resulting from both pectorales. Stoops much; occasionally squints; "snorts much in sleep"; often rejects food by nostrils. Intellect and speech reported good; "speaks in a babyish lisping manner".
XXXVIII	Charles H. H. (659.)	7	Rigid contraction of left elbow and wrist. Left tendo Achillis has been operated on for contraction in Bermuda.	Head and right-shoulder presentation; at full time; labour 3 hours. Mother healthy. Child's head lay upon right shoulder the first 9 days after birth. After this date the parents observed head to grow up straight. The feet were much swelled the second day after birth. When born was very cold and weak; was put into a hot brandy-and-water bath. Had a fit at age of 18 months; the weakness of the right leg was noticed before the fit. Parents had rubbed that limb with dry mustard a month before the fit. Walked alone at 3 years old. Looks, and is reported, intelligent.

Case No.	*Initials and Date*	*Age (Yr.)*	*Description when First Seen*	*History obtained from Parents, etc.*
XXXIX	Mary B. B. June 1860 (664.)	2¾	Spastic rigidity and debility of muscular system. Right hand severely contracted; left hand much less contracted. Lower extremities very helpless.	Crossbirth. Chin presentation. Instruments used. Child's face was lacerated. Full time. First child. Mother was frightened 3 months before confinement by severe accidental injuries to her husband. Mother, aged 40, died 4 weeks after confinement. Child was baptised immediately after birth, because not expected to live. Had threatening of a fit a week ago; never had a fit before. Uses left hand. Tries to put one foot before the other. Back very round. Head "falls" back, and often to right shoulder. Cucullaris muscles thick and contracted. Intellect reported perfect. Expression of upper part of face good. Says "uncle" and "aunt". Knows every article in Noah's Ark when asked for it. Irritable, nervous, passionate; teeth cut early, decaying rapidly, and breaking off.
XL	V. F. P. Male. In consultation with Dr. Page and Mr. Pearse, Bodmin, 1860 (665.)	9 weeks	Spastic contraction in active stage. Right hand most contracted; left hand less contracted; knees and elbows slightly contracted; thumb (right) drawn into palm; feet inclined to calcaneus; no structural shortening.	First child. Labour of 83 hours' duration; presentation natural. Child thought to be dead; very black. The mother, 6 weeks after conception, was "nearly thrown from horse". Mr. Pearse reports: Cause of difficult labour was rigidity of parts, and the infant recovered quickly from the asphyxia and the head suffered much compression. Umbilical cord was dropsical, and placenta adherent. Child's face resembles that of many idiotic children in largeness of tongue and its protrusion, and low shelving forehead. Head lozenge-shaped, projecting much backwards. Left frontal region smaller than right. Makes gurgling, choking noises. Swallows well. Peculiar prominence of sternal cartilages of right ribs. July 1861, *i.e.* 15 months later, is reported to have walked along 3 weeks ago, and to have no sign of malformation or contraction whatever. Is a large and strong child.
XLI	Jemima M. B. (666.)	15	Spastic contraction of shoulder, latiss, dorsi, pectorales, teres major and minor. Ditto of elbow, which is usually extended. Ditto of wrist, especially of flexor carpi ulnaris and radialis, also of fingers. Right side only. "Leg was similarly affected until quite recently. Still walks on toes at times." Is worse when she wills to use it.	First child. Labour tedious, 2 or 3 days. Did not cry at birth. Back of head supposed to have been injured. Child constantly convulsed first 3 or 4 days; not since. Arm first noticed to be stiff when dressing infant. Speech slightly affected. Eye ditto.
XLII	Joseph W. P. In consultation with Dr. Girdwood, 1860. (668.)	3	Spastic contraction of flexors and adductors of lower extremities Hands never affected. No atrophy. Unable to stand or walk.	First child. Child born after 6 omissions of catamenia, supposed at "10 weeks before proper time; had no nails". Labour lasted 2 days. Mother confined without assistance, because birth was "quick at the last". Was a quarter of an hour without assistance. Child did not cry until removed from mother; was black for days, certainly was so for 3 days; was thought to be dead when put into hot bath; took breast after third day; used to choke

Case No.	*Initials and Date*	*Age (Yr.)*	*Description when First Seen*	*History obtained from Parents, etc.*
				with the least thing. During first 10 days was "drawn" about the face, and "nurse used to think that he would never get breath again". Was "startlish". Had distinct convulsions from second to fifth or sixth week; also when cutting eye-teeth at age of 14 months. Seems intelligent; but mother reports he can only say "mamma, baby, and a few other words". Has lately improved. Imitates other children; crawls after ball and throws it. High prominent broad forehead. Left side of cranium less developed in parietal and occipital regions. External frontal eminences developed as often seen in children who have had hydrocephalus. Mother says head was not large at birth. Left limb reported stiffer than the right, but right is more contracted. Back round and prominent; increased when he is sitting on floor and poplitea are made to touch floor. When prone, back is straight. July 1860: legs cross. Total height, 32½ inches. From ground to top of trochanter, 14 inches. Legs therefore relatively short. Can walk round chair, holding on to it. June 1861: has picked up many words.
XLIII	Arthur S. Mr. Complin, July, 1860. (676.)	4	Contraction of adductors and flexors of lower extremities. Left hand weak. "Twists in a singular manner both wrists." More paralytic than spastic.	Fourteenth child. At seventh month "catamenia" reappeared. Labour 48 hours. Accoucheur attended, but was absent at birth. Child born with navel string about neck and legs. A hard substance, as large as another child, discharged with after-birth. Child did not cry until one hour after birth, not until navel string was severed. Child was very small. Suckled as soon as washed. During first 3 months screamed much. Can shuffle on belly; has raised himself by fire-guard. Always swallowed well; sphincters correct; speech perfect; quite intelligent; spine yields backwards in dorso-lumbar region, and to left side. September 1861: is improving. (December 1861: can now support himself almost upright against chair, and walk round it. See fig. 1.)
XLIV	Lucy W. (687.)	9	General spastic rigidity of lower extremities. Left reported to be most affected. Hands awkward, but not contracted; sews pretty well; subject to slight trembling of hands.	Born at seventh month. Midwife left the child; at first thought it was dead. No particulars of asphyxia. Took the breast the day following birth. Mother observed difficulty of separating limbs 4 weeks after the birth. Well-formed head. Slight squint. Pupils large. Intelligence a little backward; no particular faculty deficient; "has not had a chance of learning"; not been to school: reads well in Testament.
XLV	Francis W. (688.)	9	General spastic rigidity of lower limbs. Right weakest and more contracted. Hands awkward, and slightly pronated. Contraction of feet	Eighth and last child. Birth at full time, easy; was "stillborn", the cord twisted twice around neck. Remained a long time dark blue; did not cry or suck until next day; very slight, scarcely noticeable convulsions, *i.e.* clenching of hands, and closing or puckering of mouth, observed at 3 months old, always after nursing

Case No.	*Initials and Date*	*Age (Yr.)*	*Description when First Seen*	*History obtained from Parents, etc.*
			increased during last two years.	him. The first peculiarity noticed was, that head was not held erect. Mother remembers the difficulty of separating thighs when washing him. Can't sit. Left side of head appears slightly flatter, but not more than sometimes perceived in children who have had infantile paralysis. Intellect reported good, but is certainly below average. He looks intelligent. At times very intelligent in his observations. Mother never noticed deficiency, and she is a very sensible person. He has little application.
XLVI	John B. W. (689.)	5	Spastic contraction of both gastrocnemii particularly of left, much increased on locomotion. Hands awkward, and pronated to slight extent. Left side of head appears flatter, but doubtful.	First child. Mother aged 35. Rigid uterus. Labour 32 hours, at full period. Delivery by forceps, after 20 minutes' application of them. Child born with laceration at external right frontal eminence and behind ear; did not rally for 3 hours; warm bath used; no remarkable colour observed; convulsed during first fortnight. Did not suck until a month old; deglutition very difficult. Convulsion of hands was so considerable as to require padding of the palms. At 3 months old strabismus occurred in one night; no convulsion at that time. Had return of convulsion, caused by too hearty a meal, at 15 months old, which left him no worse than before. Right eye principally strabismic. It appears large; palpebræ are more open. Walks a mile or two, being led. Sphincters natural. Cheerful, tractable, timid, formerly afraid of falling backwards. Knows 20 words; cannot say alphabet. When excited is powerless and much more contracted. Is a large child.
XLVII	Z. S. Male. July 1861, Mr. Turner, Deddington. (712.)	3	Spastic rigidity of both lower extremities. Left worse. Arms and hands not affected (See fig. 2.)	Mother no particular illness or accident during gestation. First child; 7 months' child Did not cry until 36 hours after birth; was not expected to live; had no flesh on him. Slept the greater part of first 2 months. Did not take breast until 70 hours after birth. Dentition early and favourable. "At 18 months used to wake up screaming, frightened, stiffened." Mr. Turner confirms statement of premature birth between "seventh and eighth month". He never observed convulsions. Physiognomy of child when first seen suggested the complex of symptoms observed in spastic rigidity (neonatorum); seems intelligent; speech perfectly good. Formerly much confined in bowels. When first put to the ground, appeared to be more drawn up directly he touched the ground, as if he did not like it. Is irritable. After the long sleep of the first 2 months of life awoke at the slightest noise or disturbance. Parents are first cousins. A second, also a 7 months' child, died 2 hours after birth: this child looked black, but cried immediately, and was convulsed.

Abstract of Cases of Wry Neck, from Abnormal Labour, or from Asphyxia at Birth

Case No.	*Initials and Date*	*Age (Yr.)*	*Description when First Seen*	*History obtained from Parents, etc.*
XLVIII	Miss A. With Mr. Brown. Camberwell. (575.)	8	Right sterno-cleido mastoideus contracted; unyielding; 1½ inch shorter than on sound side. Vertebral column below neck apparently straight, but want of symmetry of back, chest, and loins observable; left iliaco-lumbar region more hollow. Left side of chest posteriorly more prominent.	First child. Born at full period; breech presentation; turning resorted to. Supposed to have suffered injury to neck at birth. Deformity not observed until 2 years old. Division of sterno-mastoid; mechanical extension; manipulations. Cure.
XLIX	Master J. S.	10	Wry neck.	First child. Breech presentation. Slight asphyxia at birth. Force having been used, it was supposed that neck was injured.

Abstract of Cases of Spastic Rigidity, suspected to be from Asphyxia Neonatorum

L	John E. P. In consultation with Mr. Hollis, Lewisham, 1844. (79.)	3¾	Spastic contraction of gastrocnemii, flexors of knees and adductors of thighs, not severe. Cannot walk unassisted; toes inverted during locomotion; knees separable 8 in. Imperfect volition in hands. apt to drop things; trembles. No sign of ordinary paralysis.	"Heavy fit of illness" when cutting first tooth. Some difficulty of speech; occasional strabismus. Formerly strabismus was constant. When he is pleased, he stretches hands as an infant during excitement.
LI	John T. 1949 with Dr. Marshall Hall. (309.)	2	Slight spastic contraction of lower extremities.	Convulsions, when a month old, for 1 week. Ditto for many weeks, when 3 months old. Dentition late, but unaccompanied with fits. Crowing breathing from earliest infancy until 20 months old. Right side most affected; hand has recovered. It is difficult to say whether this case should not be termed spasmo-paralysis. The spasm is slight, and he is reported to have had more marked paralysis formerly.
LII	Maude M. (440.)	7	Spastic stiffness of lower extremities; no structural shortening. Choreal (?) trembling.	Convulsions first fortnight after birth.
LIII	Cecilia R. (261.)	2½	Spastic action of gastrocnemii, excited by contact with ground. Right most affected. Right hand weaker; left-handed. Right pronators contracted. No structural shortening of gastrocnemii.	Mother confined without help. Umbilical cord tied 20 minutes afterwards. Cried incessantly for weeks after birth. Mother remarked that, as an infant, she would never lie on either side, always on back. "Always sleepless; child excitable, timid, nervous; a sudden noise, cough, sneeze, or closing of a door, throws her into a paroxysm of fear." (Well nourished; looks inclined to idiocy, but memory good. Speech good. Walks, with help, on toes. Cannot crawl. Back weak.)

Case No.	*Initials and Date*	*Age (Yr.)*	*Description when First Seen*	*History obtained from Parents, etc.*
LIV	John C. In-patient London Hospital, July, 1861. Reported by Mr. Moral Mackenzie. Resident Medical Officer.	2½	Spastic rigidity of upper and lower extremities, at times relaxing or presenting itself as gentle clonic convulsions of hands and feet. Thumbs always rigidly bent; knees crossed. Right side rather more affected. Emprosthotonos. Cries when placed on back. Tension of abdominal muscles and flexor muscles of thighs prevents flattening out of trunk. Some difficulty of deglutition. Cannot articulate. Forehead receding; flattening of parietal regions. Appears to take much notice of objects.	Is the child of gipsy parents; second child. Mother was confined at night, on a common, in February, unattended. Child was not "attended to" for 3 hours; the cold during this time was intense. The child screamed occasionally during the 3 hours it was neglected. Appears well nourished. Unusually sensitive, shown when either kindly spoken to, or reprimanded; also by the pleasure he feels on being tickled, or the intense pain he suffers on being even gently pinched. Unable to stand or crawl. [This case was diagnosed, from physiognomy and general condition, to be suffering from cerebro-spinal disturbance, induced at or soon after birth. The more active suffering indicated spinal arachnitic or myelitic michief still progressing. Was much relieved in 10 days by small doses of Hyd. c. Creta and Extr. Belladonnæ, with counter-irritation. A great part of the evil will probably prove persistent.]

Abstract of Cases of Muscular Debility or Paralysis, from Abnormal Labour, or Premature Birth, or Asphyxia Neonatorum

LV	Miss F. S. In consultation with Dr. MacIntyre, Odiham, 1847. (241.)	3½	Laxity of neck, so that head has inclined since birth to right shoulder. All the limbs feel relaxed, but no distinct paralysis, and no contraction; tendency to predominance of pronation of hands. Can stand against wall; falls if attention be abstracted. Can utter a few words, and looks intelligent. Deglutition of fluids difficult.	Dr. MacIntyre reports that birth occurred at 9 months; it was a breech case. He reached the mother's side a quarter of an hour after the birth; some difficulty had been experienced in withdrawing head; ligaments of neck and medulla were supposed by Dr. MacIntyre to have been strained. Asphyxia lasted from 1½ to 2 hours. Child was recovered from this state by the constant exertions of Dr. MacIntyre, including the use of artificial respiration. Reported June 1848: Improved in every respect, but still unable to walk. Reported November 1849: Still further improvement. No contraction, except of adductors of thighs. Walks, nurse holding hands. Stands alone at sofa. Knees cross. Intelligence developing; spells a few short words; has feeble but correct use of hands. Reported April 1852. Still uses go-cart. Is held on a pony: unable to crawl. Adductors of thighs much contracted; choreal movements occasionally observed. September 1861.—Reported to have had some attacks of convulsions soon after last report; all remedials consequently suspended. Is now unable to stand or walk. Legs contracted and inverted. Hands very unsteady, so as to incapacitate from holding anything. Articulation unintelligible except to those accumstomed to her. Swallowing of liquid difficult. She has grown much. Mental faculties sound; vision imperfect, though able to read books of large type. Amiable, cheerful disposition. Now 18 years of age.

Case No.	*Initials and Date*	*Age (Yr.)*	*Description when First Seen*	*History obtained from Parents, etc.*
LVI	Miss F. E. S. (678.)	12½	Paralytic atrophy of right half of chest, and right upper extremity. Sternum depressed. Cartilages of ribs prominent. Genua valga slight, principally of right. Two or three dorsal vertebrae slightly inclined from straight line.	Second child, only 10½ months older than the first. "Was born at eighth month; was very small, like a doll"; scarcely cried; slept greater part of first 6 (?) months. No fits or convulsions. Walked when 2½ years old. Loss of use of arm not noticed until 1 year old. Liver reported torpid. Bowels always confined. "Nervous; learns less easily than other children." Does not appear below average intelligence. Every letter well pronounced. Often sits with mouth partly open. Mother, who is very intelligent person, says she did the same.
LVII	Mary B.	2¼	Very slight general paralytic (?) weakness of muscular system (as far as I can diagnose. W. J. L.). Perhaps back somewhat weak Right knee slight genu valg. Both feet said to have been inclined to valgus on beginning to walk. Now walks very well.	Seven months or 7½ months child. Cried at birth; was weakly afterwards; sucked after third day. Inversion of one foot noticed at birth. Mother was 4 hours in labour. Liq. amnii discharged at first. Accoucheur told mother not to be surprised if child should be born dead. Presentation believed to have been natural. Has always been extremely constipated, painfully shy, sensitive, and "startlish".
LVIII	Master G. A. (229.)	3	Weakness of right upper extremity, greatest in shoulder, less of elbow, least of hand; no contraction. Scapula and entire extremity smaller.	"Cross birth."

Abstract of Case of Convulsions, from Asphyxia at Birth, followed by Paralysis.

LIX	Henry F. (626.)	11 mths.	Complete paralysis of right lower extremity. Tendency to contraction of sole and calf muscles.	Mother had severe fright 10 weeks before confinement, through another child drinking from a tea kettle. Child born at full time; large child, the sixth, the others living healthy. "Did not cry at birth, was born so quickly; one pain only; born before arrival of medical attendant. Was very dark; twice during first day turned purple, foamed at the mouth, and was convulsed." At 6 weeks old had a fit; another at 5 months old, which was followed by hemiplegia. Arm recovered in 2 days. Eldest child had convulsion when 18 months old.

Atrophy of a Hemisphere from Difficult Birth

LX	C. S. Inmate of Idiot Asylum, Earls-Wood. Autopsy by Dr. Langdon Down, M.D. London. This case was	18	Hemiplegic contraction of right arm and leg.	"Mother was of most excitable temperament. and had a very difficult confinement. Child born at full period. Instrument was used." The first born. Mother died soon afterwards. The person communicating cannot recollect whether child cried immediately after birth, what was its colour, or whether it was apparently still born. When an infant, fell off a table upon the

Case No.	*Initials and Date*	*Age (Yr.)*	*Description when First Seen*	*History obtained from Parents, etc.*
	observed by me in the asylum. Its pathological physiognomy induced me to suggest to my colleague Dr. Down an inquiry as to its history. The result is seen in the fifth column.			floor, but did not appear to suffer the least injury in consequence. Was subject to fits when a few years old. "These 'fits' consisted of a falling forward of the head so as to strike the table where it was sitting; making a great outcry at night, etc." Death caused by phthisis pulmonalis 2 years after he was first seen by me. *Autopsy.*—Head.—Atrophy of left hemisphere; old apoplexy in it; cicatrized right hemisphere; cicatrized remains of small extravasation on surface. Calvarium unsymmetrical. the right side being the larger. On removing dura mater the right half was found adherent about the vertex; between the adherent portion of the arachnoid was a mass of yellowish, tough substance, about the circumference of a shilling and a quarter of an inch in thickness; the convolutions corresponding thereto were deeply indented. The encephalon weighed 2 lb. 4½ oz. The right hemisphere of the brain much larger than the left. Along both sides of the longitudinal fissure small, white, granular bodies were noticed in the arachnoid; on the left hemisphere, about the posterior part of the middle third, was a distinct depression; corresponding thereto was a cavity, which was prolonged into the anterior lobe of the cerebrum between the cineritious portion and the corpus striatum; the outer wall had a gelatinous appearance, the inner a white, pultaceous consistence Optic thalamus of left side remarkably small and dense, having a semi-cartilaginous grating under the nail. The right half of cerebrum weighed 1 lb. 2¾ oz. The left half 11¾ oz. The rest of body was examined except the spinal column.

Post-mortem to illustrate Production of Apoplectic Capillary Congestion in Child born without Pelvic Obstruction at Birth

LXI	From Dr. W. Newman, M.D., Fulbeck, Grantham.			The wife of J. D. was confined at full time, October 1859. The tenth child. No history of fright or accident. The presentation cranial; second position (face to left sacro-iliac synchondrosis; no prolapse of umbilical cord. From the earliest accession of labour-pain until child was born nearly 12 hours elapsed. Child was born 1½ hours after rupture of the membranes, with no delay in any one stage; the child was expelled stillborn. *Post-mortem, 18 hours after examination.*—Head.—No caput saccedaneum; considerable effusion of blood over posterior and superior quarter of left parietal bone. On opening skull there was found very considerable congestion of all the superficial vessels, sinuses gorged, smaller veins distinctly mapped out. Pia mater deeply injected. Every part of brain gave evidence of most intense congestion; natural tint had given place to pinkish hue, and punctuated spots oozing blood were to be seen in every part of the brain.

Spastic Rigidity and Imbecility from Asphyxia at Birth.

Case No.	*Initials and Date*	*Age (Yr.)*	*Description when First Seen*	*History obtained from Parents, etc.*
LXII			Upwards of a year and a half ago I described to Dr. Hess, the writer of accompanying letter, the class of cases I found most commonly caused by axphyxia neonatorum. I mentioned to him that a certain class of simia-like cripples who are occasionally seen wandering about streets are sometimes cases of imbeciles with spastic rigidity from asphyxia at birth. He said he thought he recognised my picture of the disease in a man familiar to Finsbury. Soon afterwards I received from him the following letter in confirmation of my opinion.	(Copy of letter from Dr. Hess to Dr. Little) "ARTILLERY PLACE; 8. 5. 60." "MY DEAR DOCTOR, I am sorry that I have not been able to present to you the young man suffering from the consequences of asphyxia, as he has been away from here. There is no doubt about the case. I have again ascertained that he was asphyxiated for 2 hours when born, and that he has always been a weak creature, very slow in his mental development, with difficulty in speaking trembling and shaky, unable to fix his attention on a book, and a bit of a punster. I write you in order to explain my apparent negligence. With kind regards. your truly, A. HESS."

Spastic Rigidity and Imbecility from Embarrassed Breathing (?)

LXIII	Miss E. P. In consultation with Mr. Collambell, June, 1858. (579 bis.)	10½	Spastic contraction of lower extremities. Talipes equino-varus right. Talipes equino-varus left. Thighs adducted. Knees very slightly contracted.	First and only child. Born of mother about 36 years old. Born at full period; birth favourable. The infant sneezed at birth; no trouble with teething, except bilious with every tooth; was startled easily with noises; remarked at 5 years old. Is still particularly timid and nervous and childish. No fits. Walked alone about 4½ years old. Had whooping-cough and measles together mildly when 3¾ years old. Hands awkward, but no contraction. Pronounces well all letters except q and r. Very feeble intellect. June 1861.—Spends whole time with dolls and tearing up paper for paper pillows. No idea of writing. Reads a little. September 1861.—Walks without assistance of stick, and appears to have much improved in mind, since able to take more exercise. [I find this case in my journal. In appearance it is very similar to cases of spastic rigidity resulting from asphyxia neonatorum. There is, however, no other evidence of embarrassed breathing at birth than the sneezing reported. Having been a first child—mother's age at least 36 years—the umbilical cord may have suffered pressure.]

DISCUSSION

DR. BARNES said, that although not able, from his own observation, to produce any facts in confirmation or negation of Dr. Little's theory, this might be due to his not having studied the subsequent history of children in connection with the phenomena attending their birth. He was now, however, able to look back upon a considerable number of children who had been born semi-asphyxiated, in consequence of difficulty involving resort to the forceps, or turning. Many of these children he knew were healthy, and did not appear to bear any trace of the difficulties that attended their birth. He had, like most obstetricians, observed that occasionally children born with difficulty were liable to convulsions for a short time; but if these survived, they commonly did well. The difficulty there appeared to be in discussing this excellent paper, arose, no doubt, from the entire novelty and originality of the subject. Dr. Little had brought before the obstetric world new matter for inquiry of the highest interest. It was closely related to the question of the causes of still-birth—a subject, also, of which little was known, at least in this country. One reason was, that there existed no large lying-in hospitals in England, for there could be no doubt that the lying-in hospitals of the Continent lent greater opportunities for investigations of this kind than existed here. Hence Dr. Little has been obliged to look to German authors for information. He (Dr. Barnes), however, hoped that the study of the causes of still-birth would be more closely prosecuted by post-mortem examinations in this country. There was a case of which Dr. Little might be glad to avail himself. It is recorded of Samuel Johnson that "he was born almost dead, and did not cry for some time." The name of Samuel Johnson was almost synonymous with intellectual grandeur, but he was well known to be affected with certain nervous disorders which Dr. Little could better interpret than the speaker.

DR. TYLER SMITH expressed the great obligation of the Society to Dr. Little for his valuable paper. There could be no doubt the author had directed attention to an original field of observation in pointing out the injuries to which the nervous system was liable during, and immediately after, birth. Cases of early paralysis and contraction had not fallen under his (Dr. Smith's) observation, but he quite agreed with the possibility of their occurrence from the causes stated. In cases of spasm of the limbs, especially the lower extremities, shortly after birth, he had attributed the condition of the limbs to an excess of the tonic contraction of the muscles natural to the fœtus in utero, and which gradually disappeared under the influence of volition and the use of the limbs. He thought dentition the great source of paraplegia and hemiplegia in young children. The irritation of teething sometimes caused paralysis by exciting convulsions, during which the nervous centres were damaged. At other times reflex paraplegia ensued, without fits, during dentition. These forms of disease were very commonly met with, especially in hospital practice, in children from six months to two years of age. The great point was to prevent these seizures by relieving the irritation of dentition, by timely scarification of the gums, and attentions to the secretions.

DR. GIBB said he was reminded of an instance that came under his observation some years ago, but which, perhaps, hardly came within the same category as those described by the author of the paper. After a lingering labour, a child was born with spastic rigidity of all the muscles on one side of the body; in fact,

it was an instance of conjunctive hemiplegia. Suspecting that the cause existed in the brain, he was allowed to make an examination of the body of the infant, and found a clot in the substance of the brain on the side opposite to that on which the hemiplegia existed. The vessels generally were very much congested about the head, and, no doubt, had the child lived, it would have remained palsied. The case was recorded at the time in one of the medical journals.*

DR. LITTLE said he quite agreed with the President that the majority of infantile spastic and paralytic contractions arose between the ages of six months and two years from cerebro-spinal disorders, and that, perhaps, for one that depended on abnormal or premature labour there were twenty or more from other causes incidental to later life. He mentioned that, not having found any reference to the affections consequent on abnormal and premature parturition in the works of English medical writers, he had referred, with some confidence, to Shakespeare, to ascertain whether any notions on the subject were contained in his works. He said, the description of the physical character of Richard III was exactly that of an individual afflicted with one kind of deformity originated at birth.

"I that am curtailed of this fair proportion,
Cheated of feature by dissembling Nature,
Deform'd, unfinish'd, sent before my time
Into this breathing world, scarce half made up,
And that so lamely and unfashionable
That dogs bark at me as I halt by them."

In the following lines Shakespeare has used more poetic licence. The great dramatist has here probably intensified some popular notions on the subject:

"If ever he have child, abortive be it;
Prodigious and untimely brought to light.
Whose ugly and unnatural aspect
May fright the hopeful mother at the view;
And that be heir to his unhappiness."

He was convinced Shakespeare had drawn the first picture from an individual who had suffered through asphyxia at birth. He, probably, was aware of the fact mentioned by Sir Thomas More, that "the Duchess of Gloster had much ado in her travail, he (Richard III) being born the feet forward."

* *Lancet*, November 13th, 1858.